

Technical Evaluation Report

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INTRODUCTION

The mission of the Research and Technology Organisation (RTO) is to conduct and promote research and information exchange. The Human Factors and Medicine Panel (HFM) cover the fields of a) Human Performance (selection, recruitment, training, communications, teamwork, human error, cognitive performance) b) Operational Medicine (aerospace, health, safety and survival of military personnel, medical evacuation) c) Human Protection (cold, heat, hypobaric, hyperbaric, radiation etc). On the 19th and 20th May 2005, civilian and military scientists, medical officers, engineers and other personnel from NATO and Partners for Peace countries met in Amsterdam, The Netherlands for a Human Factors and Medicine Panel Specialist's meeting (HFM-126/RSM) on the "Prevention of Cold Injuries". The meeting was organised by the Human Factors and Medicine Panel in close collaboration with TNO Human Factors, The Netherlands and the Royal Netherlands Navy (RLN).

Given the extent to which cold injuries have contributed to the incapacitation of military personnel and the consequent impact on operational capability and success, the present meeting clearly contributed to both the "Operational Medicine" and "Protection" fields covered by the HFM.

BACKGROUND, SCOPE & OBJECTIVES

In times of both conflict and peace, military personnel may be required to remain in cold, wet conditions for protracted periods of time. Such exposures carry the risk of general body cooling (hypothermia) and local cooling resulting in cold injury. Depending on the nature of the cold stress, the resulting injury may be either a freezing cold injury (FCI, e.g. "frostbite") or a non-freezing cold injury (NFCI, e.g. "trench foot"). Both conditions are important for military operational capability; historically they represent the most common form of non-battle related injury in military campaigns, and they carry significant and prolonged consequences for military personnel. For example, 91,000 US Army personnel in Europe (1939 – 1945) suffered cold injuries, 67% of these were NFCI the remainder were FCI. The average period of hospitalisation for those with cold injury was 50 days and only 15% returned to active duty.

The high surface area to mass ratio of the hands and feet make them prone to cooling, and the fact that their primary source of heat, local blood flow, is virtually abolished with vasoconstriction in the cold explains why the personnel that incur a cold injury usually have injured hands and/or feet. The maintenance of peripheral blood flow is thus a critical factor in the avoidance of cold injury. Cold induced vasodilatation (CIVD) in the

Report Documentation Page				Form Approved OMB No. 0704-0188	
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1. REPORT DATE 01 MAY 2005		2. REPORT TYPE N/A		3. DATES COVERED -	
4. TITLE AND SUBTITLE Technical Evaluation Report				5a. CONTRACT NUMBER	
				5b. GRANT NUMBER	
				5c. PROGRAM ELEMENT NUMBER	
6. AUTHOR(S)				5d. PROJECT NUMBER	
				5e. TASK NUMBER	
				5f. WORK UNIT NUMBER	
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) Department of Sport and Exercise Science Institute of Biomolecular Sciences University of Portsmouth Portsmouth, PO1 2DT UK				8. PERFORMING ORGANIZATION REPORT NUMBER	
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)				10. SPONSOR/MONITOR'S ACRONYM(S)	
				11. SPONSOR/MONITOR'S REPORT NUMBER(S)	
12. DISTRIBUTION/AVAILABILITY STATEMENT Approved for public release, distribution unlimited					
13. SUPPLEMENTARY NOTES See also ADM001854, Prevention of Cold Injuries (Prevention des accidents dus au froid), The original document contains color images.					
14. ABSTRACT					
15. SUBJECT TERMS					
16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT UU	18. NUMBER OF PAGES 14	19a. NAME OF RESPONSIBLE PERSON
a. REPORT unclassified	b. ABSTRACT unclassified	c. THIS PAGE unclassified			

extremities can protect against cold injury by increasing the blood flow and therefore temperature of the extremities. The extent to which this response is “protective” by design, rather than good fortune, remains a matter of some debate.

If it can be concluded that CIVD is a reliable and robust protective response, its occurrence could be promoted by strategies such as adaptation, and the use of appropriate clothing and exercise regimes. Other approaches to prevention include the recognition of the early signs of cold injury, the use of appropriate equipment, the use of new material in clothing and equipment design and, in the light of both laboratory and epidemiological data, increasing the precision of the exposure guidelines for the avoidance of cold injuries.

The interrelationships between the clinical and research areas associated with cold injury are shown in figure 1. The topics associated with the prevention of cold injuries are examined in more detail in figure 2.

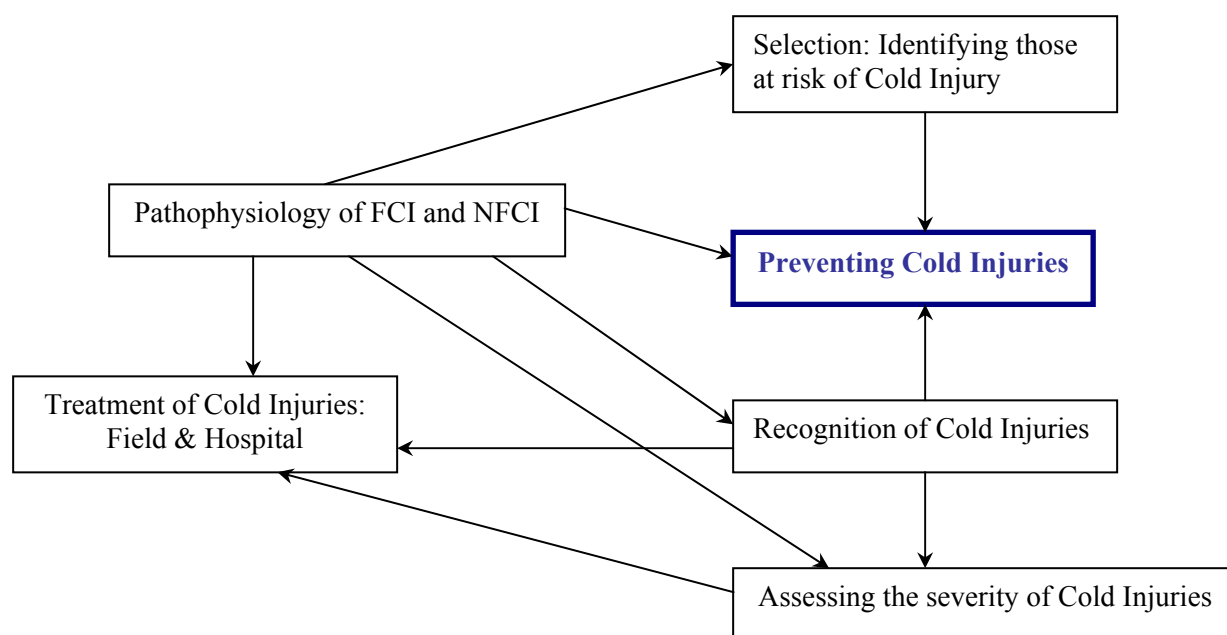


Figure 1: Clinical and Scientific Topics of Relevance to Cold Injury.

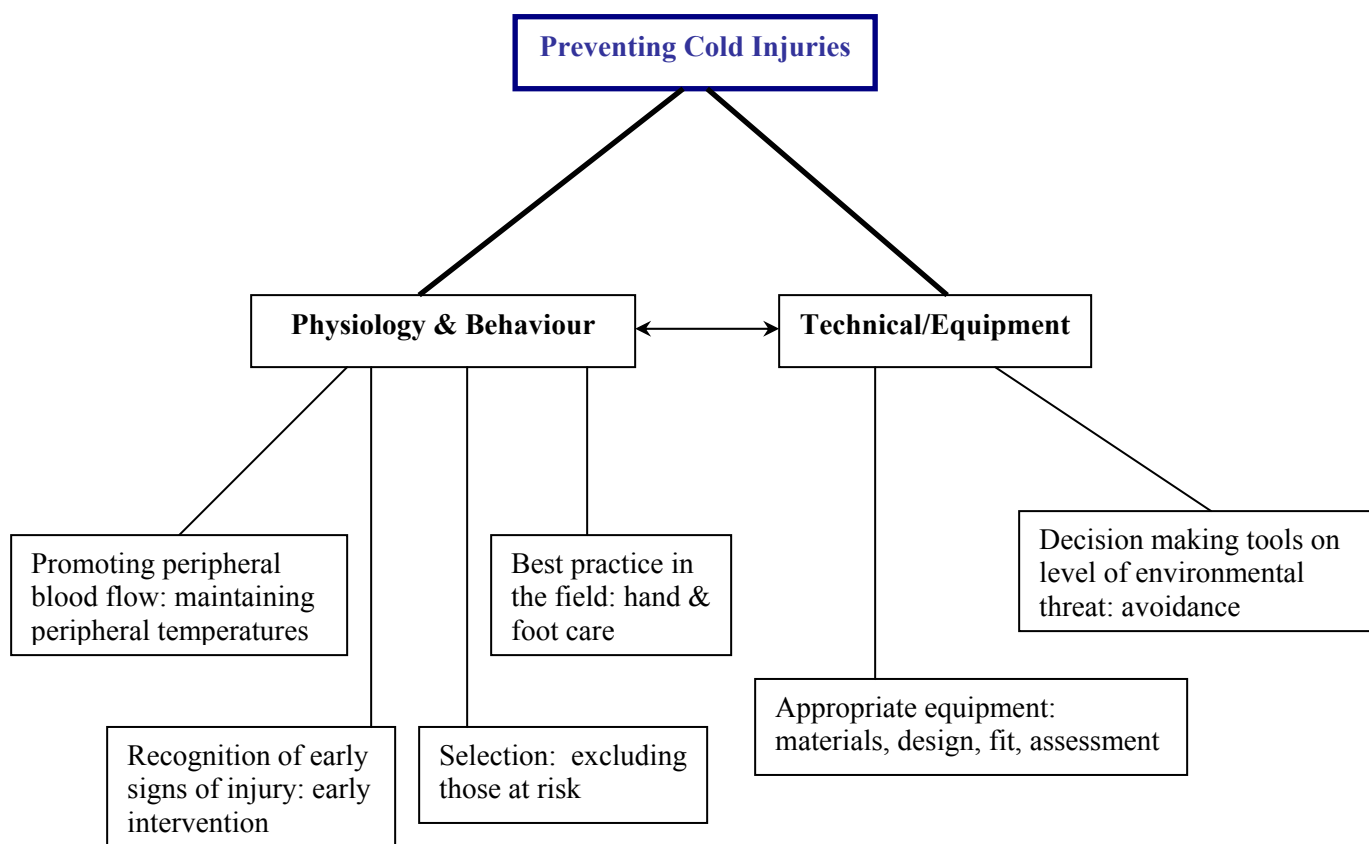


Figure 2: Topics Associated with the Prevention of Cold Injuries.

The stated objectives of the meeting were:

- i) To review the latest scientific information on protective mechanisms for cold injuries (**OBJ I**).
- ii) To develop strategies for optimal prevention (**OBJ II**).
- iii) To initiate an international database on cold induced vasodilatation and cold injury occurrence to monitor the effects of preventative measures (**OBJ III**).

THE PROGRAMME

The overall programme was chaired by Dr H. Daanen. Cdr RNLN M. Meijer from the NATO RTO in Paris attended the meeting as the HFM Panel Executive. The other members of the Programme Committee were Dr M. Ducharme from DRDC Toronto (Canada), Dr T. Makinen from the University of Oulu (Finland), Lt Col W. Korterink from the Royal Netherlands Army (The Netherlands), Dr H. Oakley from the Institute of Naval Medicine, Gosport (UK) and Cdr Dr N. van der Struijs from the Royal Netherlands Marine Corps (The Netherlands).

Dr Daanen opened the meeting and took the opportunity to emphasise the importance of the link between what scientists do and what happens in the military scenario. The welcoming address was by Vice Admiral

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van Duyvendijk, Member of the Board of Directors of TNO and former Commander in Chief of the RLN. He noted that cold injury has often been a distinctive factor in military conflict and can be experienced in relatively warm conditions. Vice Admiral van Duyvendijk thanked the sponsors and organisers of the meeting, TNO, NATO RTO and the RNN. The next speaker was Cdr Meijer who noted that 11 NATO countries were represented at the meeting and then explained a little about the current philosophy of the NATO RTO. He stated that NATO RTO have no concerns about the quality of the work being undertaken under the auspices of NATO RTO, but they would like to use some form of quality indicators of the value of the work. In this regard, Cdr Meijer then continued the theme raised by Dr Daanen, of the importance that work undertaken should be relevant to the military and address the needs of NATO. He emphasized that “Timeliness” is also part of quality and concluded with the statement “*Finis Opus Coronat*” – the end is the crown on the work.

The meeting comprised two 45 minute keynote papers, one at the beginning of each day and fifteen, 15 or 30 minute scientific presentations. The meeting was divided into five separate sessions:

- i) Risk Factors. Chaired by Dr J. Hassi and Dr M. Ducharme.
- ii) Clothing & Equipment. Chaired by Dr T. Mäkinen and Dr. E. Den Hartog.
- iii) Medical Aspects 1. Chaired by Cdr Dr G. Nobel and Dr J-B Jaquet.
- iv) Cold induced Vasodilation. Chaired by Dr J. Castellani and Dr H. Daanen.
- v) Medical Aspects 2. Chaired by Professor I. Mekjavic and Dr S. Sawada.

TECHNICAL EVALUATION

This section provides a brief review of the papers presented at the meeting, it is not intended to be a detailed exposition of each paper; the papers can easily be obtained and read individually. Rather, the salient points of each paper are listed and the objectives (OBJ I-III) of the meeting they addressed noted. A few general comments are then made about the presentations and the meeting as a whole.

Day 1 of the meeting began with the session “Risk Factors” chaired by Dr J. Hassi and Dr M. Ducharme. The first presentation was a keynote address by Dr Hassi from the University of Oulu, Finland and was entitled “Prediction and prevention of frostbite”. Dr Hassi gave a nice overview of the area and identified some of the trends that are likely to increase the chances of cold injury occurring, these include: increased urbanisation and a consequent decrease in the understanding of cold and how to deal with it; increased cold spells due to global warming; increased risk of contact frostbite in technical warfare due to the increased amount of cold metal surfaces; decreased physical fitness in military recruits. Dr Hassi gave figures for the occurrence of blister grade frostbite in the civilian population (12% in men aged 21 years, Juopperi et al, 2002; Hassi et al, 2005). In the military (peacetime), 0.4% suffer FCI in Finland (Lindholm et al 1993); Alaska 1.2 – 0.2% are medically investigated (Candler & Ivey, 1997). There was a large reduction in severe cases of FCI in Finland from 1980 – 2000, and in the US between 1985 – 1999 (de Groot et al 2003).

The important aetiological influences for cold injury include: cold/wet, immobility, posture, dehydration, inadequate nutrition, constricting footwear, fatigue, stress or anxiety, concurrent illness or injury, low fitness and the use of oily substances (Lehmuskallio, 1995), travelling in open vehicles, not wearing hats. Those with Raynauld's disease, vibration white finger and smokers should be considered for exclusion or special training (relevant questionnaire ISO 15743).

The breakout temperature for FCI has been given as: -11°C (Wilson, 1970); -4.8°C (5%) to -7.8°C (95%) (Danielson, 1996); and -18°C (Candler, 1996). The most common areas affected are the head, then hands then feet. Severe FCI is most commonly seen in the feet (Ervasti et al, 2004).

Protection: wear headgear properly adjusted, face mask, wide hood (decreases heat loss 30%); avoid oily protective smears, use watery cream before training; avoid washing. Hand protection: mittens are better than gloves; thin inner gloves; whole body thermal balance should be maintained; coating of tools to prevent/reduce cooling. Foot protection: do not stand still in bitter cold; loose fit; dry shoes; removable insoles; avoid tight socks; whole body thermal balance; avoid cold surfaces; rewarm by walking.

Longer-term problems following frostbite are common (Ervasti 2000), 63% have at least one symptom. These include sensitivity to cold, numbness and decrease tactility.

Field Commanders must take responsibility for the prevention of cold injuries; correct clothing use; prevent exhaustion; avoid sweating; avoid standing still; drink and eat enough; avoid smoking; avoid handling cold weapons; recognise warning signals (shivering, pain in fingers, numbness [Tsk <7°C]); frostnip. FCI should be recognised as a risk in the planning of activities; personnel should be trained to recognise warning signs and trained in appropriate first aid.

Dr Hassi's presentation addressed OBJ I & II of the meeting.

The next presentation was entitled "Predicting temperature limit values for cold touchable surfaces" and was given by Dr Hartog from TNO Human Factors, NL. Dr Hartog has been conducting an EU funded project to determine the maximum duration of touching and gripping materials in the cold. In four different European laboratories, six male and six female students participated in the study. All subjects touched (index finger) 10cm x 10cm blocks of wood, nylon, steel and aluminium at temperatures ranging between -40 and -5°C at three pressures (0.98, 2.94, 9.81N), for times ranging from seconds to 100s for touching, and 100 – 1000s for gripping. Thermocouples were placed on the back of the hand and the finger. The subjects scored their ratings of pain and numbness (numbness threshold 7°C) on a four-point scale. A lifting force of 500g was used for the lifting experiments.

From the cooling curve data collected, a database was developed and safety limits established for touching cold surfaces made from various materials. Modelling has allowed the group to extend the duration limits beyond the range of the data; additional modelling has revealed that the actual limits set are conservative. The new data have contributed to ISO standard NP13732 part 3 "Ergonomics of thermal environment – touching cold surfaces". Unfortunately, at this time, the group have been unable to identify a single set of parameter values to enable them to create a unique analytical model with which all conditions can be simulated; probably due to the influence of the finger thermocouple on heat exchange. This means the parameters have to be adapted to each condition (material and temperature) in order to obtain the optimal simulation of finger cooling. The data collected and analysis completed thus far suggests that the model, in addition to fitting single curves, may also be used to predict the response of the lower 25th percentile of the population. Dr Hartog concluded that the model is useful but needs to be improved; at present it is better for steady state than dynamic (initially touch) situations.

Dr Hartog's presentation addressed OBJ I & II of the meeting.

Dr Ducharme presented the paper by Ducharme & Brajkovic entitled "Guidelines on the risk and time to frostbite during exposure to cold winds".

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Dr Ducharme explained that the original wind chill index was developed for human protection: to provide guidance for clothing selection; guidance for outdoor activities; and risk assessment for development of freezing cold injuries. He then gave the rationale for revising the index, this included the fact that it was “old science”, not based on human data, it overestimated wind chill, the non-uniform use of index, and the factors not taken into account (e.g. ht measurement 10m rather than 1.5, geometry, skin insulation not considered, no correction for radiation).

The objective of the study undertaken was to define the risk and time required to develop frostbite during exposure to cold winds. Six male and six female subjects, non-smokers, age 23 – 44 years undertook sixteen 45 minute tests where the wind intensity varied between 0, 16 and 32km⁻¹. Tests were conducted at 0°C, -10°C, -20°C, -30°C, -40°C and -50°C (no wind at -50°C). Subjects were dressed for comfort and sat facing the wind with an unprotected face. Testing was terminated at 45 minutes or at the onset of frostnip.

The frequency of frostnip increased, and time to frostnip decreased with decreasing temperature. No frostnip was seen at 0°C and -10°C at any wind speed. 52 cases of frostnip were seen: 73% nose; 15% chin, 8% cheek, 4% forehead. This is comparable with the cases reported by Sipple & Passel (66% nose, 10% chin, 14% cheek, 0% forehead).

Dr Ducharme concluded that the times to develop frostbite estimated from the old wind chill index of Sipple and Passel are too short and should be revised. A new guideline based on the new Wind Chill Index is proposed, the new index was validated as described above with human subjects and with modelling. It is limited to healthy individuals, with dry bare skin, and up to 45 minutes of exposure.

Dr Ducharme’s presentation addressed OBJ II of the meeting.

Dr Geurts presented the paper of Geurts & Cheung entitled “Cardiovascular and thermal strain during manual work in cold weather”.

This work investigated the effect of cold stress and cold acclimation on neuromuscular function of the hand. A one day field study of the skin temperature and heart rate of five powerline workers (one female) working in -18°C prompted a laboratory study with 10 experimental subjects, 8 controls, to investigate the effect of physical activity, and the resulting increase in deep body temperature on cold acclimation and neuromuscular function of the hand. Neuromuscular function of the hand (tactile sensitivity, hand grip strength, manual dexterity, evoked twitch characteristics – force production from a single twitch: peak force, time to peak, half relaxation time) was assessed before and after hand cooling in 8°C water for 30 minutes while either bicycling at a submaximum level or sitting at rest, before and after two weeks of local cold acclimation (30 min/day, 5 days/week).

Neuromuscular function was impaired with cooling and neither acclimation nor an elevation in core temperature had a significant effect on manual performance. In contrast, the higher intensity of work undertaken in the field study led to the conclusion that alternating high intensity tasks with low intensity tasks demanding manual dexterity decreases cardiovascular strain and may improve manual performance by warming the body and hands.

Subjective thermal ratings were the first to acclimate even when no improvements in the temperatures measured were seen. Dr Geurts concluded that this discrepancy might represent an additional risk to those who are cold acclimated by negating their behavioural thermoregulation.

Dr Geurts' presentation highlighted an additional possible risk factor (cold acclimation) for cold injury and addressed OBJ II of the meeting.

Dr Nobel presented the paper by Nobel, Eiken, Tribukait, Kolegard & Mekjavic entitled "Motion sickness affects cold induced peripheral vasomotor tone and potentiates core cooling".

Eleven (11) healthy subjects underwent two 45 minute head out immersions in water at 15°C. Before one of the immersions subjects were made motion ill on a rotating chair, an attempt was made to maintain the level of motion illness with an rotating drum (optokinetic stimulus) mounted on the immersion tank.

When motion ill, rectal temperature decreased at a faster rate during immersion (started to fall earlier), peripheral motor tone and shivering were also attenuated. Five minutes into the immersion the motion sickness score obtained from subjects had fallen from a median score of 3 to 1; it then fell to 0.

Dr Nobel concluded that motion illness potentiates core cooling by decreasing vasoconstriction and shivering, and this may have significant implications for survival in maritime accidents.

Given that a greater degree of deep body cooling is also likely to result in lower peripheral temperatures and increase the risk of cold injury (especially non-freezing cold injury), Dr Nobels' presentation highlighted an additional possible risk factor (motion illness) for cold injury.

Professor Mekjavic presented the last paper of the morning by Mekjavic, Felicijan & Eiken, entitled, "Foot temperatures and toe blood flow during a 20km winter hike".

Ten (10) male and 10 female subjects wearing standard military winter ensemble and carrying a 20kg backpack undertook a 20km hike in an open air stadium. The ambient temperature during the 3 – 4 hour hike varied between -6°C and 8°C. The results were compared with those from subjects on guard duty (standing). Skin temperature was measured on the calf and toes; deep body temperature was measured using a gastrointestinal temperature pill (T_{gast}).

There was no change in T_{gast} during guard duty, but toe temperature fell to 15°C. T_{gast} was more variable during hiking with significant increases being observed (1 – 1.5°C), toe temperature averaged 31°C.

Professor Mekjavic concluded that those on guard duty (low activity level, immobile and upright posture) could be at risk of developing a cold injury. This risk could be minimised by using the best possible footwear, by increasing deep body temperature, or by having some form of biofeedback in the footwear.

Professor Mekjavic's presentation highlighted a risk factor (Guard duty) for cold injury and, in discussion, addressed OBJ II of the meeting.

The session on Clothing and Equipment that followed lunch was chaired by Chaired by Dr T. Mäkinen and Dr. E. Den Hartog. The first paper, by Mekjavic, Lenhart, Vrhovec, Tomsic, Bartels, Umbach, Kakitsuba, Taylor & Oakley, was presented by Professor Mekjavic.

Professor Mekjavic described the development, characteristics and capabilities of a thermal foot manikin that can be used to analyse the static and dynamic thermophysiological and mechanical properties of footwear in a range of environments, including sub-zero environments.

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Use of such a manikin should help to ensure that appropriate footwear is designed and issued for a given environment, and thereby reduce the risk of cold injury.

Professor Mekjavic's presentation addressed OBJ II of the meeting.

The next paper, entitled "Frostbite in a skiboot for marines" by Heus & Schols, was presented by Dr Heus.

Five ski boots were tested with one type of sock. Tests included:

- 1) -18°C climatic chamber, 8 subjects. 30 minutes sitting in chair, 30 minutes treadmill (5m.s^{-1}) walking, 30 minutes standing still. Temperatures on foot, weight increases in the sock, subjective sensations and body temperature were measured.
- 2) Water tightness: walking simulator in a water tank.
- 3) Water vapour transport: thermal sweating foot.

A worn ski boot had higher water vapour absorption and lower water vapour transmission than a new ski boot. Wearing a gaiter with a ski boot allowed less water vapour transmission and resulted in higher resistance to heat. None of the tested boots were waterproof, but climbing boots were better than ski boots, and new ski boots were better than worn ones. No significant differences were found between the different kind of boot combinations with regard to temperature, comfort and temperature sensations.

The greatest problem was the ski boot not being waterproof as this could increase the risk of cold injury. This problem should be addressed in a way that enables water vapour transmission.

Dr Heus's presentation addressed OBJ I of the meeting.

Dr Tomsic presented the paper by Mekjavic, Andlovec, Tomsic & Golja entitled "Phase change material in hiking boots does not minimise the risk of cold injury".

The group compared the thermal insulation properties of identical hiking boots with, or without, a layer of Outlast® phase change material (PCM). 20 males and 20 females placed their booted feet in thin plastic bags and then immersed them in water at 30°C for 15 minutes, followed by 3 hours at 15°C. Results revealed no significant difference between the boots in terms of foot skin temperature (6 sites), heat flux (6 sites) or tympanic temperature. Subjective ratings for the two conditions were also similar. Thermal insulation was $0.167 \text{ m}^2\text{K.W}^{-1}$ without the PCM and $0.163 \text{ m}^2\text{K.W}^{-1}$ with PCM.

Dr Tomsic concluded that PCM does not offer any improvement in thermal protection, and therefore does not reduce the risk of cold injury.

Dr Tomsic's presentation addressed OBJ I of the meeting.

In the final paper of the Clothing and Equipment session, Professor Mekjavic presented the paper by Mekjavic & Eiken entitled "Prevention of facial cold injury with a passive heat and moisture exchanger".

This study evaluated the thermodynamic characteristics of a prototype respiratory heat and moisture exchanger (HMS) particularly with regard to its ability to prevent cold injury to the face. The studies were conducted in a climatic chamber using a manikin attached to a respiratory simulator. Two different ventilation rates were studied (11.3 & 28L.min^{-1}) at five different ambient temperatures (-24 , -14 , -4 , 8 & 22°C). In all

subzero temperatures, the HME was able to maintain the temperature within the oro-nasal mask above 20°C. In so doing, it can effectively eliminate the risk of freezing cold injury in the oro-nasal region.

Professor Mekjavic's presentation addressed OBJ I of the meeting.

The next session was Medical Aspects 1, Chaired by Cdr Dr G. Nobel and Dr J-B Jaquet. It contained one paper by Tipton, Eglin & Golden entitled "A test for non-freezing cold injury", presented by Professor Tipton.

Having described how little is known about the pathophysiology of non-freezing cold injury (NFCI) Professor Tipton explained that one consequence of this is the difficulty in finding a definitive test to categorise the severity of the injury. Tests of peripheral blood flow are further compounded by the wide variation seen in the normal population. Indeed, in a study of 35 "normal" ("uninjured") subjects described by Professor Tipton, 28 would have been classified as having at least mild to moderate NFCI according to one test of peripheral blood flow used in a cold injuries clinic. The ideal tests of a clinical condition should be sensitive, reproducible and specific. One of the tests for NFCI conducted at the Institute of Naval Medicine (UK) requires patients to sit in a warm room (30°C) for at least 30 minutes before placing their injured limb (usually a foot) in a thin plastic bag and into 15°C water for 2 minutes. The limb is then removed and allowed to spontaneously rewarm in the warm air. The temperature of the injured part just before, and 5 minutes after, immersion is used to classify the patients. The variability, reproducibility and sensitivity associated with this test appear to be improved if a small amount of exercise is undertaken prior to the test. Stepping appears to be the best form of exercise (compared to arm cranking and cycling) and 10 minutes is enough to raise deep body temperature by about 0.3°C and, presumably, remove any central vasoconstrictor influence on what is a local injury. In the small number of patients that have been tested thus far, the incorporation of exercise in the testing regime seems to help reclassify those with moderate/severe NFCI to a less severe classification, whilst those with severe NFCI are unaffected.

Professor Tipton concluded that raising deep body temperature a little by stepping could improve the clinical test for NFCI. Furthermore, there is no "normal" rewarming response following cooling. The challenge is to decide when a response is pathological. This is similar to the relationship between blood pressure and hypertension.

Professor Tipton's presentation addressed the pathophysiology and clinical assessment of NFCI.

The second day of the meeting began with Session 4, on Cold Induced Vasodilation (CIVD). The session was chaired Dr J. Castellani and Dr H. Daanen and was opened by the keynote paper by Castellani & O'Brien entitled "Peripheral vasodilation responses to prevent local cold injuries", it was presented by Dr Castellani.

In a wide-ranging and informative review Dr Castellani focussed on CIVD, Exercise, External heat and Pre-screening as methods of avoiding cold injury. Importantly, Dr Castellani also put his evidence in the context of what would be achievable in an operational environment.

CIVD was described 75 years ago by Lewis. Various mechanisms have been suggested for CIVD including an axon reflex, decreased noradrenaline release, release of a vasodilatory substance, a cold-induced decrease in smooth muscle contractility. No definitive evidence exists for these possible mechanisms of CIVD. The CIVD response is variable, Dr Castellani addressed the questions: what defines a good CIVD responder? How are the responses defined? How large a treatment response is meaningful? What is the day-to-day reproducibility of CIVD? (O'Brien, 2005). Day to day variation: Tsk, nailbed variation 9 – 12%, and finger pad 15 – 21%. Blood flow onset and apex (time course) blood flow 5 – 9%, Tsk 18 – 24%. Having these data on variability

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enables decisions to be made on the magnitude of a CIVID change that must be observed to know if an effect has occurred; this is useful data for power analyses and provides methodological insights (variables to measure and sites to use).

Dr Castellani then explored some of the factors that modify the CIVID response, including body heat content and hypohydration (O'Brien et al, 2000; Daanen & Ducharme, 1999; O'Brien & Montain, 2003). 4% dehydration has no influence on CIVID in finger pad and nailbed. Hypoxia: CIVID occurs at lower mean finger temperature at high altitude; frostbite risk increased above 5100m. Age: CIVID later onset with age, frostbite risk higher with age. Race: Afro-Caribbeans have lower mean finger temperature and later CIVID onset, higher risk in of frostbite. Acclimatisation – enhanced CIVID, less risk of frostbite? (O'Brien & Frykman, 2003).

Importantly, given its frequent co-existence with cold injury, hypothermia suppresses CIVID such that its onset time is later with hypothermia (first vasodilatation about 6min later), finger temperature remains lower and the hunting response can disappear.

On the topic of exercise, Dr Castellani addressed the question “Does the finger respond to exercise in cold (-10°C)? (Makinen et al, 2001; Gavhed et al, 2003). The answer depends on metabolic rate. In high wind (5m/s) the increase in Tfinger with increasing work rate is much less than at slower wind velocities. Exercise increases temperatures in the fingers and face. Local acclimatisation and exercise (Stromme et al, 1963) interact; fishermen and lumberjacks react differently to students when exercising in the cold (finger temperature did not increase in the students).

Dr Castellani discussed external heating – maintaining finger and toe temperature and dexterity at -15°C. (Brajkovic et al, 1998; 2001). Torso heating is important for maintaining finger temperatures and will even work when bare handed. Without external heating fingers cool even with gloves.

Finally, Dr Castellani touched on pre-screening strategies (Smyth et al, 1999; Susol et al, 2000) before concluding that: CIVID acclimation is perhaps not practical; exercise is practical and effective, yet may not be able to be sustained; external heating is effective but power supply represents a problem; pre-screening is valuable; increased vigilance, training in the cold, education, leadership and early recognition are critical.

Dr Catellani's presentation addressed OBJ I & II of the meeting.

Dr Sawada presented his paper entitled “Assessment of local cold tolerance of individuals by using conventional and unconventional methods based on observation of CIVID reactivity”.

Dr Sawada reviewed the findings of Professor Yoshimura (1950, 1952) and others, and described the original test for CIVID that involved immersion of a finger in iced water for 30 minutes. He then described a less severe test (5 or 10°C for 10 – 15 minutes) that should represent a smaller perceptual and cardiovascular load. Dr Sawada reported that the new test correlates well with the old test and can replace it.

Dr Sawada's presentation introduced a new test for CIVID.

Dr Daanen then presented his paper entitled “The risk index for frostbite”.

Dr Daanen posed the questions “are subjects with a reduced CIVID response more prone to CI in the field?” and “is there a relationship between CIVID, smoking, ethnicity and pain in the cold?” The group studied 226 marines: 201 Caucasians, 124 non-smokers. The subjects undertook the Yoshimura protocol, in which

their left middle finger was immersed in ice water for 30 minutes. Ambient temperature was 19°C. Finger temperature (T_{finger}), pain score (0 – 5) and oral temperature were measured ($n = 94$). Minimum finger temperature, CIVD onset time and mean temp of the CIVD response (Yoshimura & Iida, 1950) were analysed. The Risk Index for Frostbite (RIF) was determined on the basis of the finger skin temperature response time and magnitude (3 = high risk; 9 = low risk). RIF results: Japanese reference = 5.7; present study = 7 (i.e. faster CIVD response); smokers 7.4; non-smokers 6.7; Caucasians 7; Non-Caucasians = 6.4 (nsd). Pain was well correlated to RIF score (10 min $r = -0.44$).

One year later, 1080 marines went to Norway, 54 received cold injuries: feet (41) hands (14), head (2). 12 of the marines were in the original RIF pool. In comparison with the other 198 marines in the RIF pool, the 12 who became cold injured had lower RIF scores (5.3 v 7.1, $P < 0.001$).

Dr Daanen concluded that the RIF shows considerable differences between subjects and, when determined in a simple laboratory test, may be related to the risk of obtaining a cold injury during operations in the field.

Dr Daanen's presentation introduced the possibility of using a test of CIVD as a screening test for risk of cold injury, and addressed OBJ II of the meeting.

The next paper was that of Ducharme, Greif, Sessler, Doufas & Mokhtarani entitled “Effect of forearm tissue temperature on the CIVD response” and was presented by Dr Ducharme.

CIVD is related to the thermal state of the body (Daanen & Ducharme 1999; Daanen et al, 1997; Bergersen et al, 1999). The group tested the hypothesis that: the influence of the thermal state of the body on the CIVD response could be attributed to a peripheral effect caused by changes in the perfusion characteristic of the tissues proximal to the CIVD site.

On two different occasions 11 healthy male subjects preconditioned their forearm tissues at two different water temperatures (20°C and 38°C) until steady state forearm temperature was achieved. Measures: T_{sk} (7 sites), $T_{\text{sk forearm}}$, T_{ty} , $T_{\text{sk tip of fingers}}$, T_{muscle} (3cm in *flexor digitorum profundus*), $T_{\text{blood radial artery}}$. Following pre-conditioning, the fingers of the conditioned forearm were placed in a water bath at 5°C for 30 minutes in order to examine the CIVD response. During the immersions in the different conditions skin and deep body temperature did not differ, but temperature 3cm into the forearm was different (23.6°C v. 36.7°C in 20°C and 38°C preconditioning respectively). T_{blood} was cooler at the wrist (28.2°C) with 20°C pre-conditioning than (35.6°C) with 38°C pre-conditioning.

Forearm pre-conditioning caused significant differences in all CIVD parameters during the 30 minute immersion in 5°C water. Notably, CIVD onset time was slower (7.8s) with 20°C pre-conditioning than 38°C (5.2s); Peak time slower (11.7s v 5.9s) and peak amplitude lower (2.1s v 4.4s) with 20°C preconditioning.

Dr Ducharme concluded that low forearm tissue temperature impedes CIVD, despite normal body skin and core temperatures; possibly due to a decreased arterial blood temperature or flow to the skin (it could also be due to a central effect mediated by afferents from the forearm muscles). Pre-conditioning the forearm may decrease risk of cold injury.

Dr Ducharme's presentation addressed OBJ I of the meeting.

The final session of the meeting on Medical Aspects 2 was chaired by Professor I. Mekjavic and Dr S. Sawada. The paper by Jaquet, Hovius, Daanen & Brandsma entitled “Prevention of cold injuries: what can be learned from nerve injury patients” was presented by Dr J-B Jaquet.

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Patients with upper extremity nerve injury often complain about cold intolerance, reduced sensitivity and impaired manual performance. The prognosis following nerve injury is not that good. (40 – 45% “good” recovery). Motor recovery takes 3.5 years, sensory 4 years. 5.5 years after surgery a close relationship has been found between sensory recovery and level of cold intolerance.

A pilot assessment of the sensibility of soldiers with frostbite showed them to have diminished sensation. The group then investigated the effect of sensory recovery on CIRD and cold intolerance. 136 subjects (107 responded, 85 male) participated with ulnar, median and combined injuries. Eight of the cold intolerant subjects immersed their hands in 15°C water for 5 minutes, after which infrared pictures of their affected hands were taken. The damaged regions could most easily be identified 5 minutes after immersion. Median nerve injury removed CIRD from the fingers served by the median nerve (1, 2, 3); CIRD remained in fingers 4 & 5 – served by the ulnar nerve (opposite happens with ulnar nerve lesion).

Dr Jaquet concluded that the comparison between cold injury and nerve injury patients might yield interesting information about the nervous involvement in NCCI. He further concluded that the infrared temperature profile of the damaged hand after cold water immersion may be a helpful tool to assess nerve damage.

Dr Jaquet’s presentation addressed the pathophysiology and clinical assessment of NCCI.

The final paper of the meeting was that of Mekjavic, Gorjanc, Mekjavic, Bajrovic & Milcinski. It was entitled “Hyperbaric oxygen as an adjunct treatment of freezing cold injury” and was presented by Dr Gorjanc and Professor Milcinski.

With the exception of freezing cold injury, hyperbaric oxygen therapy (HBOT) is now accepted as an adjunct therapy for the management of all other acute traumatic peripheral ischaemias (e.g. crush injuries, compartment syndrome, thermal burns, compromised skin grafts). This paper tested the hypothesis that HBOT may help in the treatment of cold injury.

The paper presented the experience gained from treating 16 frostbitten patients with HBO. All were experienced alpinists who received field medical attention on returning to base camp and, within a few days to a week, HBOT (2.5ATA, 90 minutes, 100% O₂ for 25 minutes via an oro-nasal mask/5 minute break, repeated 3 times – treatments twice a week). The number of HBOT ranged from 11 – 30. Bone scintigraphy was performed prior to HBOT on 7 patients, as this appears to have a positive predictive value for amputation.

The results suggested that the progression of recovery and results of HBOT are largely dependent on the time between injury and the onset of HBOT, the magnitude of the injury and the field treatment. In general, oedema subsided by the 3rd to 5th day of HBOT, and revitalisation of the affected region was evident on the 2nd day of HBOT. HBOT also caused the demarcation line to move distally. Additionally, Dr Gorjanc noted that the predictive value of the new French grading system for freezing cold injury is valid; early first aid is important; and scintigraphy adds important information and predicts lesions.

Dr Gorjanc concluded that more data are required to determine the optimal HBOT, but treatment should be started as soon as possible. Although control data are hard to come by, accepting that frostbite is, in part, due to anoxia of the tissues, means that HBOT should be considered as an adjunct therapy.

Dr Gorjanc and Professor Milcinski’s presentation addressed the treatment of freezing cold injury.

GENERAL COMMENTS, CONCLUSIONS & RECOMMENDATIONS

The organisers of the meeting, and those that supported it, should be congratulated for producing a successful meeting that brought together many of those working in the field of cold injuries.

The objectives of the meeting were:

- i) To review the latest scientific information on protective mechanisms for cold injuries (**OBJ I**).
- ii) To develop strategies for optimal prevention (**OBJ II**).
- iii) To initiate an international database on cold induced vasodilatation and cold injury occurrence to monitor the effects of preventative measures (**OBJ III**).

Objective I and II were addressed by the papers presented at the meeting, Objective III was not. However, this objective is important and should be supported. Some of the papers presented did not directly address any of the objectives of the meeting, dealing instead with: risk factors for cold injury; the clinical assessment and treatment of cold injury; and new tests for NFCI and CIVD. Although strictly outside the remit of a meeting on the “Prevention of Cold Injuries”, these papers made an important contribution. This raises the question of whether the meeting was too narrow in its focus, and whether the other topics shown in figure 1 could also have been included. To some extent they were, by default, by the inclusion of the papers just highlighted. It can be seen from figure 1 that, of all of the topics shown, it is an understanding of the pathophysiology of cold injury that has the greatest impact on the other topics within the area. This is particularly the case for NFCI. Despite the fact that it accounts for the majority of cold injuries, that it has been a problem for centuries and has characteristics that are similar to other conditions with peripheral vascular pathology, we still do not understand the pathophysiology of NFCI. It is this gap in our knowledge that contributes to the difficulties we have identifying susceptible individuals, as well as the problems associated with recognising and assessing NFCI, and treating it in the field and hospital. Research into the pathophysiology of NFCI should be encouraged. This is unlikely to be short-term or inexpensive, but a thorough understanding of this condition will have significant implications for operational capability.

Cold injuries are of significant military operational importance and the military should be giving serious consideration to this condition. In this context, it would have been nice to see more operational active military personnel present at the meeting. The scientists can help achieve this by ensuring that their research is relevant to the military and reported in a way that makes its impact on operational capability clear. This point echoes that made by Dr Daanen and Cdr Meijer at the start of the meeting. Some of the papers presented at the meeting had room for improvement in this regard. Military personnel and scientists must remain in close contact: critically the military liaison must be with someone with operational experience; this is the best way to ensure that scientifically underpinned advice is provided to the commanders in the field in a relevant format. The responsibility does not all lay with the scientist, the military must use their scientific support. They must also be prepared to fund and support some longer term projects that have clear operational significance (e.g. that mentioned above on the pathophysiology of NFCI).

It was clear from the meeting that a good deal of work has been undertaken on CIVD and its relationship with cold injury. It should be noted however, that many peripheral cold injuries occur coincidentally with general hypothermia: to be protective in these cases CIVD would need to occur in individuals with lowered deep body temperatures. On balance the published evidence does not support this conclusion. The work examining the relationship between the CIVD and cases of cold injury incurred in the field, which is currently in its early stages, should be supported and extended. This should establish if individuals with a reduced CIVD response are at greater risk of cold injury. The association between these two factors may be coincidental, or operating

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via an unknown third factor, but still be of sufficient strength for CIVD to be used as an important screening tool.

Operationally relevant work should continue in the areas highlighted in figures 1 & 2. It is recommended that particular attention be given to:

- Knowledge transfer from the laboratory to the field: the best mechanism and format for getting scientifically underpinned advice to the end users in the field.
- Knowledge transfer from the field to the laboratory: more than ever before data can be collect remotely. This should enable relevant assessment of clothing and equipment, as well as help elucidate the aetiology of cold injury.
- Identification of the benefits and insights to be gained from other associated fields (e.g. peripheral neuropathies).
- The causes, identification and treatment of cold injury:
 - Models for the aetiology and pathophysiology of FCI and NFCI.
 - Biochemical, physiological and genetic markers of cold injury.
 - Sensitive, reproducible and specific tests for identifying those at risk in the “normal” population.
 - Protocols for treatment in the field, when to medivac and best practice for treatment in hospital and subsequently.

ACKNOWLEDGEMENTS

Thanks to the organisers for inviting me to act as Technical Evaluator for this meeting, and to Tara Reilly for her help and hard work.